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Citation for published version (APA):

Verhoef, S. P., Camps, S. G., Gonnissen, H. K., Westerterp, K. R., & Westerterp-Plantenga, M. S. (2013). Concomitant changes in sleep duration and body weight and body composition during weight loss and 3-mo weight maintenance. *American Journal of Clinical Nutrition*, 98(1), 25-31. <https://doi.org/10.3945/ajcn.112.054650>

Document status and date:

Published: 01/07/2013

DOI:

[10.3945/ajcn.112.054650](https://doi.org/10.3945/ajcn.112.054650)

Document Version:

Publisher's PDF, also known as Version of record

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Concomitant changes in sleep duration and body weight and body composition during weight loss and 3-mo weight maintenance^{1–3}

Sanne PM Verhoef, Stefan GJA Camps, Hanne KJ Gonnissen, Klaas R Westerterp, and Margriet S Westerterp-Plantenga

ABSTRACT

Background: An inverse relation between sleep duration and body mass index (BMI) has been shown.

Objective: We assessed the relation between changes in sleep duration and changes in body weight and body composition during weight loss.

Design: A total of 98 healthy subjects (25 men), aged 20–50 y and with BMI (in kg/m²) from 28 to 35, followed a 2-mo very-low-energy diet that was followed by a 10-mo period of weight maintenance. Body weight, body composition (measured by using deuterium dilution and air-displacement plethysmography), eating behavior (measured by using a 3-factor eating questionnaire), physical activity (measured by using the validated Baecke's questionnaire), and sleep (estimated by using a questionnaire with the Epworth Sleepiness Scale) were assessed before and immediately after weight loss and 3- and 10-mo follow-ups.

Results: The average weight loss was 10% after 2 mo of dieting and 9% and 6% after 3- and 10-mo follow-ups, respectively. Daytime sleepiness and time to fall asleep decreased during weight loss. Short (≤ 7 h) and average (> 7 to < 9 h) sleepers increased their sleep duration, whereas sleep duration in long sleepers (≥ 9 h) did not change significantly during weight loss. This change in sleep duration was concomitantly negatively correlated with the change in BMI during weight loss and after the 3-mo follow-up and with the change in fat mass after the 3-mo follow-up.

Conclusions: Sleep duration benefits from weight loss or vice versa. Successful weight loss, loss of body fat, and 3-mo weight maintenance in short and average sleepers are underscored by an increase in sleep duration or vice versa. This trial was registered at clinicaltrials.gov as NCT01015508. *Am J Clin Nutr* 2013;98:25–31.

INTRODUCTION

Parallel to the increase in the prevalence of obesity, a reduction in sleep duration has been observed in the past decades (1, 2). Evidence has been accumulating that short sleep duration is a risk factor for weight gain (3, 4). Consequently, it has been suggested to add sleep duration to a growing panel of determinants that contribute to obesity (3).

Sleep deprivation may predispose individuals to obesity via energy intake, energy expenditure, and substrate use (5–9). Short sleep duration can increase energy intake through an alteration in the neuroendocrine control of appetite and reward (6, 7, 9–12). Moreover, short sleep duration increases opportunities to eat, and in combination with highly available palatable foods,

this can lead to an increase in energy intake (13). Energy expenditure can be decreased with short sleep duration by a decline in physical activity caused by an increased tiredness during waking hours (5) or by an altered thermogenesis (14). Although some studies have shown an effect of short sleep duration on specific components of energy metabolism such as activity energy expenditure (7), the total daily energy expenditure does not seem to be affected (7, 15). Stress also may play a role through the relation between the activity of the hypothalamic-pituitary-adrenal axis, insulin sensitivity, and substrate use (16).

The relatively short- or medium-term observational studies may elucidate mechanisms that supposedly induce continuous weight gain, but whether they lead to the development of obesity in the long term remains questionable. Therefore, it is necessary to observe whether sleep duration and body weight, including body composition, are related to each other and change concomitantly over the longer term. A few studies have addressed this research question. Rat studies have shown a quantitative relation between sleep and the energy status of the body (17, 18) and a qualitative relation between sleep and lipid-protein synthesis and degradation (17). Nedeltcheva et al (14) showed that a combined energy and sleep restriction in humans resulted in a decreased loss of fat mass and increased loss of fat-free mass. These effects suggest that sleep plays a role in the preservation of human fat-free mass during periods of energy restriction. Another long-term study showed that shifting sleep duration from a short to a healthier length is associated with lower adiposity gain (19). Sleep duration and quality at baseline were both associated with body fat loss in a moderate caloric-restriction intervention (20).

The studies have suggested an inverse relation between sleep duration and the development of body weight. However, it is impossible to ascertain the temporal sequence from case-control and cross-sectional studies because they assess sleep duration and body weight concurrently. Only longitudinal study designs are suitable to investigate whether changes in sleep duration are followed by changes in body weight or vice versa.

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² No funding was received for this study.

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Received November 12, 2012. Accepted for publication April 10, 2013.

First published online May 22, 2013; doi: 10.3945/ajcn.112.054650.

The purpose of this study was to assess the relation between changes in sleep duration and changes in body weight and body composition during weight loss in the long term. More specifically, we investigated whether a temporal sequence of changes in sleep duration and body weight and body composition could be revealed.

SUBJECTS AND METHODS

Subjects

A total of 150 healthy subjects aged 20–50 y with BMI (kg/m^2) of 28–35 were recruited by using advertisements in local newspapers and on notice boards at the university. Subjects underwent a screening and were in good health, nonsmokers, not using any medication (except for oral contraception), and moderate alcohol users. None of the subjects gained or lost >5 kg in the 3 mo before the study. Ninety-eight subjects (25 men and 73 women) completed the study. The weight-loss diet consisted of an 8-wk very-low-energy diet (VLED) that provided 2.1 MJ/d (Modifast; Nutrition et Santé Benelux). This diet was a protein-enriched formula diet that provided 50 g carbohydrates, 52 g protein, 7 g fat, and a micronutrient content, which met the Dutch recommended daily allowance. Vegetables were allowed in addition to the diet. The weight-loss period was followed by a weight-maintenance period of 10 mo, in which subjects were instructed to maintain their newly achieved body weight without specific dietary instructions. Measurements were performed at 4 time points before weight loss, after weight loss, and after 3- and 10-mo follow-ups. The study was conducted according to the guidelines laid down in the Declaration of Helsinki, and all procedures involving human subjects were approved by the Central Committee on Human Research and the Medical Ethical Committee of the University of Maastricht. Written informed consent was obtained from all subjects. This trial was registered at clinicaltrials.gov as NCT01015508.

Anthropometric measures

Height was measured at screening to the nearest 0.1 cm with the use of a wall-mounted stadiometer (model 220; Seca). Body weight was measured with subjects wearing underwear after an overnight fast by using a calibrated scale. BMI was calculated by dividing body weight by height squared. The distribution of fat was investigated by measuring the waist circumference at the site of the smallest circumference between the rib cage and the iliac crest with subjects in a standing position. Hip circumference was measured at the site of the largest circumference between waist and thighs. Body composition was calculated from body volume with the use of air-displacement plethysmography (BodPod; Life Measurement) (21) and total body water (22) as assessed by using the deuterium-dilution technique with the use of Siri's 3-compartment model (23). The dilution of the deuterium isotope ($^2\text{H}_2\text{O}$) is a measure for total body water. Subjects wore tightly fitting bathing suits and a swim cap during volume measurements of the air-displacement plethysmography and had not engaged in exercise ≥ 1 h before the test.

Blood variables

Fasted blood samples were taken and collected in EDTA-containing tubes to prevent clotting. Plasma was obtained by

centrifugation and stored at -80°C until additional analysis. Leptin concentrations were measured by using the human radioimmunoassay kit (Millipore).

Questionnaires

Subject characteristics were assessed at all visits through specific questionnaires. To determine whether the attitude toward food intake changed during weight loss and follow-up, a validated Dutch translation of the 3-factor eating questionnaire was used (24). To determine physical activity, the validated Baecke's questionnaire was used (24, 25).

For sleep duration, questions were "How many hours do you usually sleep per night during weekdays?" and "How many hours do you usually sleep per night during weekend days?" A total weekly sleep score was calculated as follows (5):

$$\frac{[(\text{Hours of sleep on weekdays} \times 5) + (\text{hours of sleep on weekend days} \times 2)]}{7} \quad (1)$$

Another question concerning sleep quality was "How long does it take to fall asleep?" Daytime sleepiness was assessed by using the Epworth Sleepiness Scale. Subjects rated the likelihood of falling asleep in 8 specific situations on a 0–3 scale, with 0 meaning no chance at all of falling asleep and 3 meaning a high chance of falling asleep. The total score could range from 0 to 24, with a score of 10 and higher suggesting excessive daytime sleepiness (26).

Statistical analysis

Data are presented as means (\pm SDs) unless otherwise indicated. A repeated-measures ANOVA was carried out to determine possible differences over time. Age and sex were used as covariates in all tests. Because subjects consisted of short sleepers (≤ 7 h sleep), average sleepers (>7 to <9 h sleep), and long sleepers (≥ 9 h sleep) (3), changes in sleep duration were assessed for short and average sleepers separately from changes in long sleepers. Linear regressions were carried out to determine relations between dependent and independent variables. Significance was defined as $P < 0.05$. All of statistical analyses were executed with SPSS software (version 16.0 for Macintosh OS X; SPSS Inc).

RESULTS

A total of 98 subjects (25 men and 73 women) completed the study (**Figure 1**), with an average weight loss of 10% after the VLED and 9% after the 3-mo follow-up and 6% after the 10-mo follow-up compared with at baseline. Body weight, BMI, fat mass, and hip circumference were decreased compared with at baseline (**Table 1**). Also leptin concentrations decreased (**Table 1**), with a significant positive correlation between the change in leptin and the change in fat mass after 10-mo follow-up ($P < 0.01$, $R^2 = 0.32$). Dietary restraint scores increased, and disinhibition scores decreased (**Table 1**). The change in dietary restraint scores was negatively correlated with the change in BMI and fat mass after the 10-mo follow-up ($P < 0.01$, $R^2 = 0.17$ and $P < 0.01$, $R^2 = 0.15$, respectively). The change in disinhibition scores was positively correlated with the change in BMI and fat

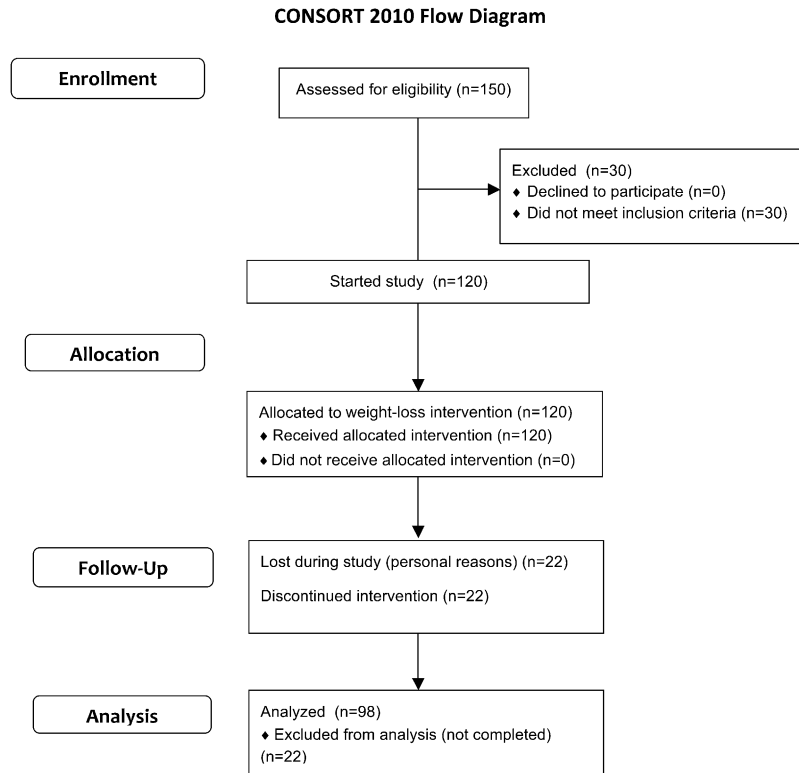


FIGURE 1. Flow diagram (CONSORT). CONSORT, Consolidated Standards of Reporting Trials.

mass after the 10-mo follow-up ($P < 0.05$, $R^2 = 0.03$ and $P < 0.05$, $R^2 = 0.03$, respectively). There were no significant differences in the percentage of fat mass, waist circumference, hunger scores, and physical activity levels over time after correcting for sex and age. The Epworth Sleepiness Scale was decreased (Table 1), which indicated a decrease in daytime sleepiness. Furthermore, the time to fall asleep slightly but significantly decreased (Table 1).

In short and average sleepers, sleep duration increased during weight loss (0.4 ± 0.7 h; $P < 0.01$). To assess whether this change in sleep duration was associated with changes in body weight and body composition, linear regression analyses were performed. The change in sleep duration was negatively correlated with the change in BMI after weight loss and after the 3-mo follow-up (Figure 2). After the 10-mo follow-up, there was no significant correlation between the change in sleep duration

TABLE 1
Subject characteristics over time at t0, t2, t5, and t12 ($n = 98$)[†]

	t0	t2	t5	t12	P
Body weight (kg)	92.5 ± 12.7	83.0 ± 11.3*	84.4 ± 12.4*	86.8 ± 13.2	<0.001
BMI (kg/m ²)	31.9 ± 3.2	28.7 ± 3.1*	29.1 ± 3.4*	29.9 ± 3.7	<0.001
Fat mass (kg)	38.7 ± 7.6	31.2 ± 7.5*	30.9 ± 8.3	33.6 ± 8.6	0.021
Percentage of fat mass	41.8 ± 6.6	37.5 ± 7.3	36.5 ± 7.7	38.5 ± 6.8	0.196
Waist circumference (cm)	96.9 ± 9.6	89.4 ± 8.7	89.7 ± 9.3	94.2 ± 11.3	0.089
Hip circumference (cm)	112.7 ± 7.5	106.1 ± 7.5*	106.3 ± 7.7	108.6 ± 9.0	0.010
Leptin concentration (μg/L)	27.5 ± 16.0	13.5 ± 12.2*	21.4 ± 18.5*	21.0 ± 16.2	0.004
Dietary restraint score	7.1 ± 3.7	12.6 ± 4.3*	12.1 ± 4.4*	11.4 ± 4.4*	<0.001
Disinhibition score	6.4 ± 2.8	4.9 ± 2.7*	5.3 ± 2.9*	5.7 ± 2.7*	0.010
Hunger score	5.2 ± 3.0	3.7 ± 3.0	3.4 ± 2.9	3.9 ± 2.9	0.098
Work activity score	2.6 ± 0.6	2.6 ± 0.6	2.7 ± 0.7	2.7 ± 0.7	0.401
Sport activity score	2.3 ± 0.7	2.4 ± 0.6	2.5 ± 0.7	2.4 ± 0.7	0.255
Leisure activity score	2.9 ± 0.6	3.0 ± 0.6	3.0 ± 0.6	3.0 ± 0.6	0.624
Sleep duration (h)					
Short and average sleepers	7.6 ± 0.9	8.0 ± 1.0*	7.8 ± 1.0	7.8 ± 0.9	0.005
Long sleepers	9.6 ± 0.9	8.9 ± 1.3	8.8 ± 1.0	8.7 ± 1.3	0.375
ESS	5.5 ± 3.3	5.2 ± 3.4	4.8 ± 3.4	5.0 ± 3.5*	0.036
Time to fall asleep (min)	18.8 ± 17.0	15.5 ± 14.9	16.6 ± 15.8	17.1 ± 16.6*	0.009

[†] All values are means ± SDs. *P* values are for differences over time and were determined by using repeated-measures ANOVA. *Significantly different from baseline ($P < 0.05$). ESS, Epworth Sleepiness Scale; t0, baseline; t2, after the very-low-energy diet; t5, after the 3-mo follow-up; t12, after the 10-mo follow-up.

and BMI. To assess the temporal sequence, a linear regression analysis was performed between the change in sleep duration during weight loss and the change in BMI after follow-up. These correlations were not significant after the 3- or 10-mo follow-up. Also, vice versa, the change in BMI during weight loss was not correlated with the change in sleep duration after follow-up, which indicated that changes in BMI and in sleep duration only occurred in parallel during weight loss.

To assess whether the change in sleep duration in short and average sleepers was associated with the change in fat mass, linear regression analyses were also performed with the change in fat mass. During weight loss, this correlation was not significant. At the 3-mo follow-up, the change in sleep duration was negatively correlated with the change in fat mass (**Figure 3**). After the 10-mo follow-up, there was no significant correlation between the change in sleep duration and the change in fat mass. As for BMI and fat mass, there was no significant correlation between the change in sleep duration during weight loss and the change in fat mass after follow-up or vice versa. Again, this result indicated that changes in fat mass and sleep duration occurred in parallel until 3 mo after weight loss. A multiple regression analysis of changes in sleep duration and changes in BMI, respectively, showed that fat mass did not increase the explained variation of changes in BMI alone. However, changes in body weight and fat mass both after weight loss and after the 10-mo follow-up were not significantly different between groups.

Change in sleep duration was inversely correlated with sleep duration at baseline in the entire group (**Figure 4**). With a closer inspection of data, it appeared that the variance in sleep duration had decreased remarkably after weight loss. Short and average sleepers increased their sleep duration during weight loss [0.7 ± 1.1 h ($n = 21$; $P < 0.01$) and 0.2 ± 0.5 h ($n = 57$; $P < 0.01$), respectively]. Sleep duration in long sleepers did not significantly

change during weight loss after the inclusion of age and sex as covariates ($n = 20$; -0.7 ± 1.0 h; $P = 0.37$).

DISCUSSION

The average weight loss was 10% after 2 mo of dieting ($P < 0.01$), 9% after the 3-mo follow-up ($P < 0.01$), and a non-significant 6% after the 10-mo follow-up ($P = 0.05$). Sleep duration increased in short and average sleepers during weight loss. This change in sleep duration was concomitantly negatively correlated with the change in body weight during weight loss and after the 3-mo follow-up and with the change in fat mass after the 3-mo follow-up. Short (≤ 7 h) and average (>7 to <9 h) sleepers increased their sleep duration during weight loss, whereas the sleep duration in long sleepers (≥ 9 h) did not change significantly. These results suggested that sleep duration in short and average sleepers benefits from weight loss and vice versa.

The inverse correlations between changes in sleep duration and changes in BMI indicated that successful weight loss and maintenance occurs concomitantly with an increase in sleep duration. The slope coefficient of the correlation between changes after the 3-mo follow-up (time 5 – time 0) was higher than the slope coefficient of the correlation between changes after weight loss (time 2 – time 0) ($\beta_{2-0} = -0.5$; $\beta_{5-0} = -0.7$). Changes in BMI and sleep duration appeared to be the largest during weight loss, and they stabilized during the 3-mo follow-up. However, after the 10-mo follow-up, there was no longer a significant correlation between the increase in sleep duration and decrease in BMI. This result might have been because of the larger variation in weight maintenance, with some subjects who regained their lost weight and some subjects who lost even more weight, whereas the newly achieved sleep duration was maintained overall. To assess whether an increase in sleep duration would precede a decrease in BMI or

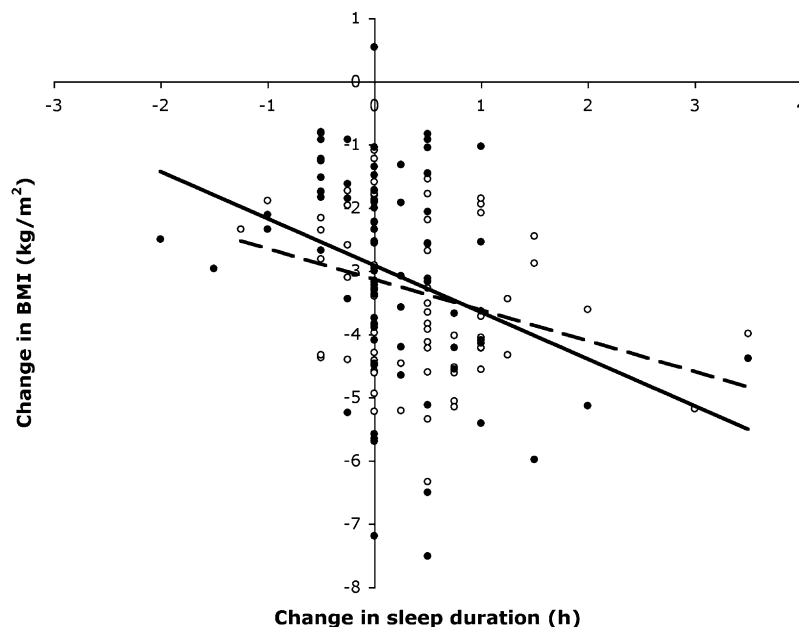


FIGURE 2. Changes in BMI (in kg/m^2) as a function of the change in sleep duration (h) during weight loss (changes over $t2-0$: white circles and dotted trend line) and after the 3-mo follow-up (changes over $t5-0$: black circles and black trend line) in short and average sleepers ($n = 78$). Linear regression BMI_{t2-0} compared with $\text{sleep duration}_{t2-0}$: $P < 0.05$, $R^2 = 0.09$ ($y = -0.4857 \times -3.137$) and BMI_{t5-0} compared with $\text{sleep duration}_{t5-0}$: $P < 0.01$, $R^2 = 0.10$ ($y = -0.7411 \times -2.9135$). Changes over $t12-0$ were not significantly correlated and, therefore, are not depicted. $t2-0$, period of weight loss between baseline and after 2 mo of weight loss; $t5-0$, period between baseline and after 3-mo follow-up; $t12-0$, period between baseline and after 10-mo follow-up.

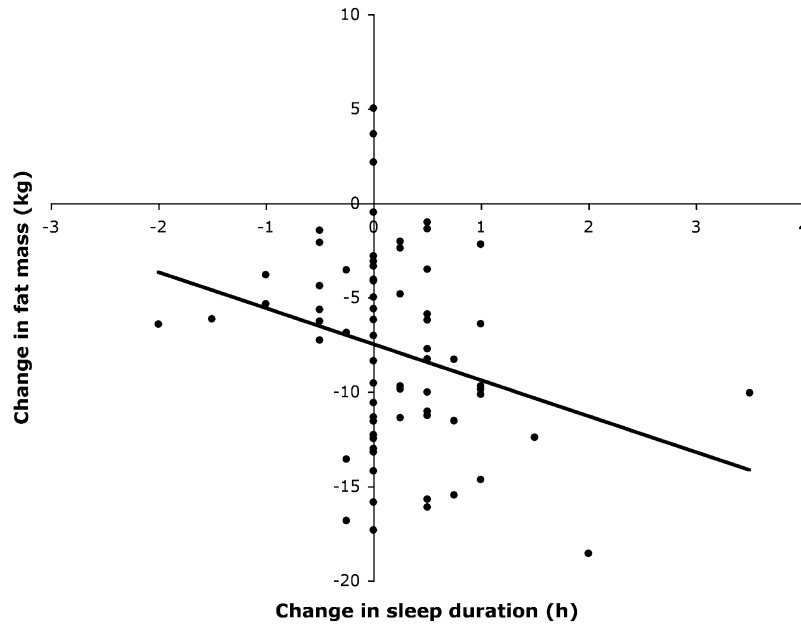


FIGURE 3. Changes in fat mass (kg) as a function of the change in sleep duration (h) after the 3-mo follow-up (changes over t5–0: black circles and black trend line) in short and average sleepers ($n = 78$). Linear regression fat mass_{t5–0} compared with sleep duration_{t5–0}: $P < 0.05$, $R^2 = 0.07$ ($y = -1.906 \times -7.4817$). Changes over t2–0 and t12–0 were not significantly correlated and, therefore, are not depicted. t2–0, period of weight loss between baseline and after 2 mo of weight loss; t5–0, period between baseline and after 3-mo follow-up; t12–0, period between baseline and after 10-mo follow-up.

vice versa, the possible relation between the change in sleep duration during weight loss and change in BMI during follow-up was tested in a linear regression and vice versa. No significant correlations were shown, and therefore, no conclusion could be drawn on the temporal sequence. Longitudinal cohort studies suggested that short sleep duration is associated with future weight gain (27, 28). However, the first study could not be generalized because the study included persons with high risk of psychiatric disorders (27), whereas the other study could not exclude the possibility

of reverse causation because only sleep duration was assessed at one time point (28).

Moreover, it is impossible to disentangle cause and effect because of different study designs (29, 30). To our knowledge, only effects of sleep restriction have been investigated because, until now, it has not been known how to intervene by using sleep improvement, whereas changes in body weight mainly have been shown by using diet-induced weight loss. To our knowledge, no studies have shown spontaneous changes in body weight and

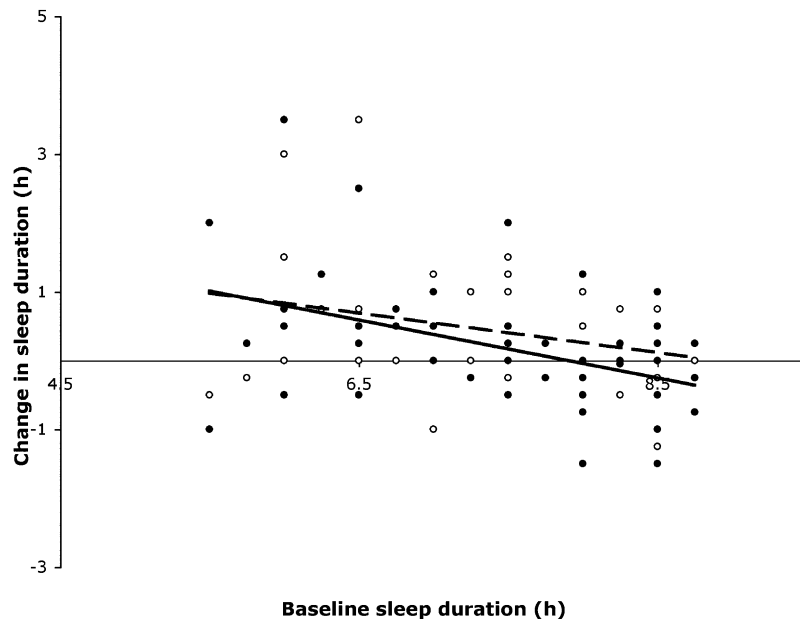


FIGURE 4. Changes in sleep duration (h) as a function of baseline sleep duration (h) in short and average sleepers ($n = 78$) (changes over t2–0: white circles and dotted trend line; changes over t12–0: black circles and black trend line). Linear regression sleep duration_{t2–0} compared with sleep duration_{t0}: $P < 0.01$, $R^2 = 0.11$ ($y = -0.2876 \times +2.5597$) and sleep duration_{t12–0} compared with sleep duration_{t0}: $P < 0.001$, $R^2 = 0.21$ ($y = -0.4204 \times +3.3209$). Changes over t5–0 were not significantly correlated and, therefore, are not depicted. t0, baseline; t2–0, period of weight loss between baseline and after 2 mo of weight loss; t5–0, period between baseline and after 3-mo follow-up; t12–0, period between baseline and after 10-mo follow-up.

sleep duration at the same time. From the currently available studies, it seems that parallel changes in sleep duration and BMI may have a common underlying cause, which can exert its effect in a parallel manner. For example, stress might act on sleep duration and body weight independently and possibly exert a synergistic effect.

The second objective of this study was to assess whether an increase in sleep duration from short to normal sleep duration would be associated with a larger decrease in fat mass. Nedeltcheva et al (14) showed that a combined energy and sleep restriction in humans resulted in an increased loss of fat-free mass. This result suggested that sleep plays a role in the preservation of human fat-free mass during periods of energy restriction. However, no constructive conclusion can be drawn from studies that restricted both energy intake and sleep duration as in the study of Nedeltcheva et al (14). Therefore, we assessed spontaneous changes in sleep duration during both energy restriction and a weight-maintenance period. The inverse correlation between the change in sleep duration and change in fat mass after the 3-mo follow-up suggested that, in addition to a concomitant correlation between a change in sleep duration and weight loss and weight maintenance, body fat loss was also concomitantly associated with a change in sleep duration or vice versa. However, the addition of the change in fat mass in a multiple regression did not strengthen the association between the change in BMI and change in sleep duration. Thus, the association of the change in sleep duration with the change in fat mass might be included in the association with the change in BMI. Changes in body weight and fat mass during follow-up were not significantly different between groups, which indicated that sleep-duration habits did not influence body weight stability in the reduced obese state.

During weight loss, sleep quality was improved with a decrease in daytime sleepiness and a decrease in the time to fall asleep. Previous studies have shown that there is an interaction between sleep quality and the lipid-protein synthesis and degradation (17). Minet-Ringuet et al (18) showed that sleep quality improved by refeeding rats with α -lactalbumin. Such studies suggested that adjustments in the diet could be used to improve sleep in adults and may also improve body weight regulation (31).

Sleep duration increased in short and average sleepers during weight loss, whereas sleep duration in long sleepers did not change significantly. These results suggested that sleep duration in short and average sleepers benefits from weight loss and vice versa. Chaput et al (19) concluded that shifting sleep duration from a short to healthier length is associated with lower adiposity gain. However, it is still unclear what this healthier sleep duration would be. For clinical implications, it is important to find the optimum sleep duration and by which factors it is determined to be able to use this information in combination with changes in diet and exercise to optimize weight loss.

A limitation of this study was the use of self-reported sleep durations, although previous studies showed good agreement between self-reported and measured sleep durations (32, 33). The time to fall asleep significantly decreased; however, a decrease of only 1–2 min might not have been clinically relevant. After 10-mo follow-up, many anthropometric variables (ie, body weight, BMI, fat mass, and waist and hip circumference) had returned to baseline values in some of the subjects, and thus,

these variables lost their significance with respect to the difference shown at the 3-mo follow-up. This result was probably because of a large variation in the body weight regain of subjects, which may have indicated a transient effect of the negative energy balance during the VLED in some of the subjects. During weight loss, subjects were allowed to eat vegetables in addition to the diet, and results indicated that subjects, during weight loss, consumed a total of 5.2 MJ/d, including the diet. In addition, effects of potential behavioral and metabolic changes induced by the VLED of only 5.2 MJ/d itself could not be excluded.

To our knowledge, this study was differentiated from previous studies because of its longitudinal design to investigate the relation between spontaneous changes in sleep duration and changes in body weight and body composition as a result of energy restriction.

In conclusion, sleep duration benefits from weight loss or vice versa. Successful weight loss, loss of body fat, and 3-mo weight maintenance in short and average sleepers are underscored by an increase in sleep duration or vice versa.

We thank Loek Wouters and Jos Stegen for their assistance.

The authors' responsibilities were as follows—KRW and SPMV: designed the study; SPMV and SGJAC: collected data; SPMV: analyzed data and wrote the manuscript; MSW-P and HKJG: contributed to the interpretation of data and reviewed the manuscript; KRW: supervised the execution of the study; and all authors: read and approved the final manuscript. None of the authors had a conflict of interest.

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